Pneumonia Associated with Near-Drowning

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Drowning and near-drowning can abruptly devastate the lives of both the affected victims and their families. In addition to the complications directly caused by the submersion, several indirect causes of morbidity exist. Infection is one of the complications associated with near-drowning, and pneumonia is the most severe of these infectious complications. The risk factors, microbiological causes, diagnostic approach, and appropriate therapy for pneumonia associated with near-drowning are not well described in the literature. Herein, we review the epidemiology and pathophysiology associated with near-drowning, discuss the potential mechanisms of infection, and describe the likely risk factors for pneumonia related to near-drowning. We also detail the microbiological causes of this entity and provide important clinical and epidemiological information associated with specific pathogens. Finally, we summarize an appropriate diagnostic and therapeutic approach to pneumonia associated with near-drowning.

Drowning and near-drowning are catastrophic events that occur unexpectedly in the lives of individuals who are often young and otherwise healthy. Common mechanisms for injury are hypoxemia, acidosis, and hypoperfusion. While pulmonary and neurological damage is a major cause of morbidity and mortality, a submersion event can be devastating to all of the major organ systems. Other life-threatening abnormalities include cardiovascular instability and renal failure due to ischemia and metabolic abnormalities and disseminated intravascular coagulation [1, 2]. Infection is yet another potential life-threatening complication of near-drowning. While the attributable morbidity and mortality associated with infection in victims of near-drowning are not known, bacterial and fungal infections certainly play a role. Of the infections associated with near-drowning, pneumonia is one of the most devastating. We review the epidemiology and pathophysiology of drowning and near-drowning and relate them to near-drowning-associated pneumonia. We also summarize the pathogens that have been described as causes of near-drowning-associated pneumonia and suggest an appropriate diagnostic and therapeutic approach for patients with near-drowning-associated pneumonia.

Epidemiology

Incidence. The annual rate of submersion injuries is unknown [2], primarily because of incomplete reporting. The confusing and sometimes inconsistent terminology used to describe immersion events also impacts on the estimated incidence of these tragedies. The following terms are used in describing submersion events [3]. *Drowning* is death from asphyxia within 24 hours of submersion. *Near-drowning* is a submersion episode of sufficient severity to warrant medical attention. *Secondary drowning* is death from complications >24 hours after the submersion event. *Immersion syndrome* is sudden death, probably mediated through the vagus nerve, due to cardiac arrest after contact with cold water. *Submersion injury* is any submersion resulting in hospital admission or death (e.g., near-drowning, drowning, or spinal cord injury). A *save* is a water rescue or removal of a victim from water by someone who perceived the individual to be a potential victim of a submersion injury.

Drowning causes ~8,000 deaths per year in the United States, making it the fourth leading cause of accidental death. Near-drowning is estimated to occur from 2 to 20 times more frequently [2–4]. The percentage of near-drowning victims who develop pneumonia is unknown, as most of the information available on this subject is based on anecdotal case reports and series. Near-drowning victims have not yet been prospectively assessed for the frequency of this complication. Kennedy et al. [5] prospectively studied children who had experienced severe aspiration events. Thirteen individuals in that prospective group were near-drowning victims. Seven of those near-drowning victims developed infection (probably pneumonia, although this was not explicitly described). Unfortunately, the true incidence of infection after submersion cannot be inferred from this study because patients whose blood was not cultured in the first 48 hours and those who survived <24 hours were excluded.

Oakes et al. [6] retrospectively reported 16 episodes of pulmonary infection in 40 near-drowning victims, but their review did not provide diagnostic criteria for these cases. Modell et al. [7] mentioned only one individual with pneumonia in their

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retrospective review of 91 near-drowning victims; however, they did not include the complication of pneumonia as part of the analysis. In addition, episodes of pneumonia following submersion have been described in multiple small case series. Thus, while the incidence of pneumonia associated with near-drowning is unknown, the findings in these series suggest that it is not a rare event.

Age, sex, and comorbid conditions. Most drowning and near-drowning involves males, particularly those in the 10–19-year age group [2, 3, 8]. This factor is probably related to the high-risk activities that males engage in during their adolescent years. Consequently, near-drowning-associated pneumonia has been more commonly reported for males. There is no reason to expect males to be at more risk biologically for pneumonia after a near-drowning. For females, the highest rate of drowning and near-drowning is among those 1–2 years old, reflecting a known propensity of toddlers for accidents. The peak incidence of drowning and near-drowning is bimodal for males, with the highest incidences observed during toddlerhood and adolescence, reflecting both the predisposition of toddlers for accidents and the high-risk behavior more commonly displayed by young men [3, 8].

The majority of drowning and near-drowning incidents involve young and otherwise-healthy individuals. Drowning is the third most common cause of death overall in the 10–19-year-old age group, second only to motor vehicle injuries as a cause of accidental death [3]. For this young, healthy population, a relatively good rate of recovery from near-drowning-associated pneumonia would be expected. Contrary to this assumption, however, the case fatality rate associated with near-drowning-associated pneumonia is 60% (30 deaths in 50 cases) (table 1). Pneumonia caused by *Aeromonas* species, *Burkholderia pseudomallei,* and *Pseudallescheria boydii* after near-drowning appears particularly morbid [9–17]. The relatively high case fatality rate associated with near-drowning pneumonia due to these organisms is somewhat unexpected because the victims have been young and otherwise healthy. The mortality attributable to pneumonia among these individuals is unknown, and other factors related to the near-drowning may certainly have played significant roles.

Pathophysiology

The ultimate mechanism of injury after all submersion events is hypoxemia- and ischemia-induced organ damage [2, 18–20]. However, the events leading to the final outcome are varied and have an unclear impact on the risk of near-drowning pneumonia. In order to accurately determine the risk of pneumonia associated with near-drowning (as it relates to pathophysiological mechanisms), a prospective trial must be done. However, several factors associated with the pathophysiology of near-drowning likely determine the probability that pneumonia will develop.

Aspiration. One factor that likely determines risk of pneumonia after near-drowning is whether the victim aspirates. Following an unexpected submersion, voluntary apnea occurs. The apnea continues until the $PaCO_2$ and PaO_2 levels reach a threshold beyond which involuntary breathing occurs, resulting in aspiration of water. Some individuals will aspirate a small amount of fluid during the initial apneic phase resulting in laryngospasm. In many of these victims, the laryngospasm will eventually relax due to hypoxemia, allowing further aspiration. However, for unclear reasons, the laryngospasm will not resolve in a small minority of drowning victims, and they will drown without aspirating fluid. Approximately 10% of drowning victims do not aspirate [1, 3, 4, 7]. By extrapolating this information to near-drowning victims, it would be expected that a significant minority of these individuals would avoid aspiration and possibly be at lower risk for complicating pneumonia. Most near-drowning victims aspirate <3–4 mL/kg of fluid, and postmortem studies of drowning have shown that at least 85% of victims aspirate <22 mL/kg [2, 21]. The volume of aspirate may influence the risk of pneumonia after a submersion event. One would intuitively expect a larger inoculum to place the victim at higher risk of infection, but this supposition has never been studied.

Primary or secondary event. Another pathophysiological factor that may impact on the risk of developing near-drowning pneumonia is the secondary nature of many of these submersion events. A significant number of submersion events are secondary to seizures, syncope, trauma, or sudden death [20]. These individuals often do not have a voluntary apneic phase and may aspirate substantially more fluid than other near-drowning victims. However, one could postulate that reflex laryngospasm is still present in these victims, allowing for substantial protection from large-volume aspiration. Whether suffering near drowning as a secondary event increases an individual's risk of pneumonia is unknown.

Gastric aspiration. The aspiration of gastric contents as a result of near drowning may also increase the risk for near-drowning-associated pneumonia. While aspiration of large volumes of fluid is uncommon in near-drowning incidents, gastric ingestion of large volumes of fluid is not [3, 5, 8]. Often individuals in the panic of an unexpected submersion event swallow a significant amount of water. This places the near-drowning victim at significant risk for vomiting, particularly during resuscitation. With the victim often neurologically compromised, aspiration of vomitus easily occurs. Autopsy studies have shown that approximately one-fourth of drowning victims aspirate vomitus [20]. Aspiration of gastric contents causes significant inflammatory damage to the pulmonary parenchyma, which places the person at increased risk of pulmonary infection, not only with oropharyngeal organisms, but also with those found in the aquatic environment.

Contaminated aspiration. Several other factors may play a role in determining the risk of near-drowning-associated

Table 1. Characteristics of near-drowning-associated pneumonia caused by specific organisms.

Organism	No. of cases: Definite*	Probable†	Possible‡	Case fatality rate	Unique epidemiology or comorbid condition(s) (no. of patients)	Time of near-drowning to pneumonia (no. of patients)	Comments (no. of patients)§
Aerobic gram-negative bacteria							
Aeromonas species	8	3	0	7/11	Cirrhosis (1)	Within 24 h (8)	Bacteremia (8)
Burkholderia pseudomallei	6	0	0	5/6	Location, Southeast Asia (5)	All illnesses occurred within 2 w	Lung cavity (1); miliary (4) and focal consolidation (1); bacteremia (4)
Chromobacterium violaceum	0	2	2	0/3	Location, Florida (3)	Delayed onset, >1 mo (3/4)	Hepatic abscess (3); bacteremia (3)
Francisella philomiragia	0	3	2	1/5	. . .	Within 5 days (5)	Bacteremia (5)
Klebsiella pneumoniae	0	0	1	0/1	. . .	Within 4 days (1)	. . .
Legionella species	0	3	0	2/3	Chronic lymphocytic leukemia (1)	Within 4 days (2); delayed onset, 6 w (1)	. . .
Neisseria mucosa	0	1	0	1/1	Alcoholism (1)	Within 24 hours (1)	Bacteremia (1)
Pseudomonas aeruginosa	2	2	0	2/4	Near-drowning in hot tub (3)	Within 5 days (4)	Bacteremia (2)
Shewanella putrefaciens	0	0	1	0/1		Within 4 days (1)	. . .
Vibrio species	0	0	2	2/2		Within 4 days (2)	Bacteremia (1)
Aerobic gram-positive bacteria							
Streptococcus pneumoniae	3	0	2	3/3	All <11 y of age	Within 24 h (3)	Bacteremia (5)
Staphylococcus aureus	0	0	1	?			
Fungi							
Aspergillus species	0	1	0	0/1		Within 7 days (1)	. . .
Pseudallescheria boydii	5	4	1	8/10	Chronic liver disease (1); diabetes mellitus (1)	Onset delayed >1 mo (3)	Disseminated infection, most commonly CNS (9)

* Definite = chest radiographic evidence of pneumonia or histopathologic evidence of pneumonia and a positive culture of a specimen from a normally sterile site (blood, pleural fluid, or CSF) and a positive culture of an airway specimen (same organism isolated from the sterile sight).

† Probable = chest radiographic evidence of pneumonia or histopathologic evidence of pneumonia and a positive culture of a specimen from a normally sterile site (blood, pleural fluid, or CSF) or a positive culture of an airway specimen.

‡ Possible = chest radiographic evidence of pneumonia or histopathologic evidence of pneumonia or a positive culture of a specimen from a normally sterile site (blood, pleural fluid, or CSF) or a positive culture of an airway specimen.

§ Denominator reflects number of patients for which information was available.

pneumonia. A submersion event in contaminated water (e.g., that containing sewage or manure) likely increases the risk of pneumonia. The types and amounts of human pathogens found in the aquatic environment are significantly increased in contaminated water. Aspiration of contaminated water increases the risk of pneumonia due to certain organisms, as described below. Seventy percent of drowning victims aspirate mud, sand, and fragments of aquatic vegetation [20]. As a result, the surrounding environment may play a significant role in the risk and type of near-drowning-associated pneumonia. Furthermore, individuals who experience near-drowning in a shallow environment are more likely to aspirate particulate material, potentially increasing the risk of near-drowning-associated pneumonia after shallow-water submersion events.

Water temperature. The temperature of the water involved in a submersion event may affect both the likelihood of pneumonia and the type of organism causing illness. The metabolic and division rate of organisms generally increase with increasing temperature [22]. Thus, the number of organisms increases with increasing temperature. While this concept is not true in all situations (e.g., polluted water), it is usually the case. In general, bacteria and fungi have a narrow temperature range in which they can survive. Psychrophilic bacteria (those that grow optimally in a cold environment), such as *Vibrio marinus,* as a rule are unable to survive at temperatures >25°C [22]. Because these bacteria predominate in cold-water environments and generally do not tolerate human physiological temperatures, pneumonia following very-cold-water submersion events is probably less common. This point is not trivial, as Alaska has the highest drowning rate of any state in the United States, primarily due to occupational exposure [8]. The lack of case reports of near-drowning-associated pneumonia from the state of Alaska, despite the high number of submersion events, suggests a lower rate of this complication following cold-water submersions.

Chemical composition. The chemical composition of the water certainly affects the type of pathogenic organism involved in the pulmonary infection, but it is unknown if this factor affects the overall rate of infection. Factors such as salinity, turbidity, pH, and the presence of dissolved gases, organic substances, and inorganic substances all affect the number and type of organisms in the environment [22]. It might be expected that a saline environment is more conducive to the growth of pathogenic organisms that could thrive in a human physiological environment. However, seawater is ~3.5% saline, a percentage that is significantly higher than that (0.9%) in human blood [23]. Many marine bacteria experience osmotic lysis when exposed to water with lower salt concentrations [22]. Most microorganisms grow only within a rather narrow range of salt concentration (1%–5%). Furthermore, experimentally induced aspiration of saltwater and freshwater in dogs reveals no significant difference in terms of physiological effects [24]. While more case reports of near-drowning-associated pneumonia involve freshwater events, this finding may reflect the fact that more immersion events occur in freshwater [3, 8].

Nosocomial pneumonia. Finally, while near-drowning certainly elevates the risk of pneumonia caused by waterborne organisms and typical oropharyngeal organisms, individuals who experience near-drowning are also at significant risk for nosocomial pneumonia. The significant damage done to the epithelial lining of the lungs places these individuals at higher risk for developing pulmonary infection. Many of these victims require monitoring in the intensive care unit, mechanical ventilation, and H_2 blockers. These factors increase the risk of nosocomial pneumonia, most commonly due to gram-negative aerobic organisms [25].

Table 2. Type of environmental exposure related to organisms causing near-drowning-associated pneumonia.

Organism	Freshwater	Saltwater	Contaminated, stagnant water
Aerobic gram-negative bacteria			
Aeromonas species	+++	+	+
Burkholderia pseudomallei	++		+
Chromobacterium violaceum	++		++
Francisella philomiragia	?	++	
Klebsiella pneumoniae		+	
Legionella species	+		
Neisseria mucosa		+	
Pseudomonas aeruginosa	+	?	++
Shewanella putrefaciens		+	
Vibrio species	?	+	
Aerobic gram-positive bacteria			
Streptococcus pneumoniae	++	+	
Staphylococcus aureus	?	?	
Fungi			
Aspergillus species	?	+	+
Pseudallescheria boydii	?	?	+++

NOTE. Data are as follows: +++ = frequently reported; ++ = occasionally reported; + = rarely reported; and ? = Potential cause of illness on the basis of environmental isolation.

Organisms that Cause Near-Drowning-Associated Pneumonia

The causes of pulmonary infection associated with near drowning are quite varied. Most of the organisms causing this syndrome are only described in case reports or small case series. Consequently, no single case definition is used to describe near-drowning-associated pneumonia, and some cases reviewed in the literature may not represent true pulmonary infection. Defining near-drowning-associated pneumonia is particularly difficult, as many victims develop adult respiratory distress syndrome, and some develop fever without clear infection. Despite these limitations, both bacteria and fungi have been well-described in the literature as causes of pulmonary infection after a submersion event. The following section reviews the organisms which cause pneumonia after near-drowning and the setting in which these organisms are likely to cause this syndrome (tables 1 and 2).

Aerobic gram-negative bacteria. Aeromonas species are prevalent causes of pneumonia associated with near-drowning [9, 26–33]. *Aeromonas hydrophila* is the most common aeromonad reported to cause near-drowning-associated pneumonia, but this finding may represent a failure to differentiate species in the clinical laboratory until recently [9]. The organism is found in both freshwater and brackish water and causes pneumonia after near-drowning in both saltwater and freshwater. While invasive infection with *Aeromonas* species is more commonly reported among individuals with liver disease and hematopoietic malignancies, most episodes of near-drowning-associated pneumonia due to this organism occur in otherwise healthy hosts [9]. More than 70% of these victims have bacteremia. The mortality associated with this serious aeromonas infection may approach 60%, although the attributable mortality is unknown. Most of the cases that have been described involved the development of sepsis and pneumonia within 24 hours of admission to the hospital after a brief period of stability after submersion. While the antimicrobial susceptibilities of *Aeromonas* depend on the species, most aeromonads are susceptible to aminoglycosides, extended-spectrum penicillins, third-generation cephalosporins, carbapenems, aztreonam, trimethoprim-sulfamethaxazole, and fluoroquinolones [34].

Burkholderia pseudomallei is the organism that causes melioidosis, a disease that occurs almost exclusively between 20° north latitude and 20° south latitude and is endemic in Southeast Asia [35]. The organism can be isolated from soil and freshwater in Southeast Asia [36, 37]. *B. pseudomallei* is also a cause of near-drowning-associated pneumonia in areas where this pathogen is endemic [10, 11, 38]. A 46-year-old female with no reported medical illness developed *B. pseudomallei* pneumonia 2 weeks after nearly drowning in a river near Manila, the Philippines [37]. The patient developed a cavitary lung lesion, and both sputum and blood cultures yielded *B. pseudomallei.* The patient recovered after she was treated with cephalothin and amikacin.

Four military personnel in South Vietnam developed submersion-associated pneumonia with *B. pseudomallei* after being propelled into a rice patty by an explosion [10]. All of these patients sustained significant burns but were clinically stable until ~1 week into their hospitalizations, when they developed septic shock. Three of the patients died within 48 hours, and autopsies revealed that *B. pseudomallei* involved multiple organs, including the lungs. One patient was treated presumptively with chloramphenicol and tetracycline. He initially responded to treatment but subsequently died. It is unclear in these cases if the pathogen entered through the respiratory tree because of aspiration or because of breaks in the integument caused by the burns these patients had sustained.

A 30-year-old healthy Thai man developed *B. pseudomallei* pneumonia and sepsis after he was involved in a motor vehicle accident and aspirated muddy water [11]. Cultures of blood, sputum, and the environment at the accident site yielded *B. pseudomallei.* The patient died of complications of his illness. Most *B. pseudomallei* are susceptible to trimethoprim-sulfamethoxazole, third-generation cephalosporins, extended-spectrum penicillins, carbapenems, monobactams, chloramphenicol, and tetracyclines [39–41].

Chromobacterium violaceum is a common soil and freshwater inhabitant found in subtropical and tropical climates that rarely causes human illness. Most infections in the United States occur in the Southeast, particularly in Florida [42]. Several cases of *C. violaceum* infection are described in association with exposure to stagnant water [42, 43]. *C. violaceum* is also associated with near-drowning-associated pneumonia.

Four cases of *C. violaceum* sepsis associated with near-drowning have been described in the literature [42, 44–46]. In three of the four cases, the illness occurred >1 month after the near-drowning. Bacteremia occurred in three of the four cases, and pulmonary infiltrates were present on chest radiographs in two of four cases. Positive sputum cultures or autopsy cultures of the lung were not described in any of these cases. *C. violaceum* pneumonia associated with near-drowning may involve a different pathophysiology than other causes of near-drowning-associated pneumonia. As infection with *C. violaceum* often causes disseminated disease (including involvement of the lungs) and several cases have occurred after exposure of wounds to water [42, 43], the portal of entry is likely through the skin, with resultant bacteremia and eventual development of hepatic and pulmonary infection and abscesses. This concept is also supported by the significant delay in the development of pneumonia and the fact that positive sputum cultures have not been reported. Clinicians should suspect this pathogen when a patient appears septic up to several weeks after a near-drowning in stagnant water in the southeastern United States. The organism is usually susceptible to extended-spectrum penicillins, aminoglycosides, tetracyclines, chloramphenicol, trimethoprim-sulfamethaxazole, and fluoroquinolones [47]. Its susceptibility to extended-spectrum cephalosporins is variable.

Francisella philomiragia is an organism rarely reported to cause human infection. Five cases of *F. philomiragia* infection after near-drowning are reported in the literature; all occurred after immersion in saltwater [48]. *Francisella tularensis,* the organism that causes tularemia, has been isolated in freshwater and is known to cause illness associated with exposure to freshwater [49]; however, this organism is not reported to cause near-drowning-associated pneumonia.

All five of the individuals with *F. philomiragia* infection associated with near-drowning had positive blood cultures, and three had evidence of pneumonia (all of these patients had received corticosteroids within 24 hours of admission to the hospital). Three of the four patients for whom adequate information was available recovered. The nonsurvivor had severe anoxic brain damage and was declared brain dead after receiving a 7-day course of antibiotic therapy. An autopsy of this patient revealed pneumonia and pulmonary abscesses. Clinicians should suspect this organism as a cause of pneumonia in victims of near-drowning in saltwater. Although the pathogenicity of *F. philomiragia* in cases of freshwater drowning is unknown, the organism has been isolated in freshwater [50]. *F. philomiragia* is susceptible to extended-spectrum cephalosporins, fluoroquinolones, aminoglycosides, chloramphenicol, and tetracyclines [48].

Several Enterobacteriaceae, including *Klebsiella pneumoniae,* have been reported to cause pneumonia after near-drowning [5, 6, 51, 52]. Many of these reported cases of near-drowning-associated pneumonia are probably nosocomial, although classification is difficult, since few environmental cultures from the near-drowning site were obtained. *K. pneumoniae* is commonly found in environmental sites including seawater [23]. Unfortunately, while several cases of near-drowning-associated pneumonia due to Enterobacteriaceae (including *K. pneumoniae*) have been reported in limited detail [5, 6, 52], only one case of klebsiella pneumonia after a near-drowning has been described in detail [51]. A 60-year-old female developed bilateral pneumonia 20 hours after admission for a saltwater near-drowning. The patient later developed fever, and her sputum culture yielded *K. pneumoniae.* She responded well to cephaloridine and polymyxin and was discharged without further treatment 16 days after admission.

Organisms in the genus *Legionella* are known freshwater inhabitants [53, 54]. Hence, these organisms would also be expected to be a potential cause of near-drowning-associated pneumonia. A 71-year-old Japanese female developed *Legionella pneumophila* pneumonia 4 days after nearly drowning in a hot-spring spa [55]. The organism was isolated from a tracheal sample and from the site of the near-drowning. The patient was treated with erythromycin and recovered after prolonged hospitalization. One case of non-*pneumophila* legionella pneumonia was reported in a near-drowning victim [56]. The victim was a 60-year-old male with chronic lymphocytic leukemia who developed right-upper-lobe pneumonia 2–3 weeks after being submerged in dirty swamp water. Despite treatment with penicillin and cephalothin and the subsequent addition of erythromycin, the patient developed complications of his illness and died. Postmortem cultures of the lung yielded *Staphylococcus aureus, Enterococcus faecalis, Escherichia coli, Enterobacter* species, and *Bacillus* species. Culture of the lung tissue also yielded a *Legionella*-like organism. In another case suggestive of *Legionella* as a cause of near-drowning-associated pneumonia, a 36-year-old male developed bilateral bronchopneumonia 2 days after learning scuba diving in a freshwater swimming pool [57]. Despite treatment with penicillin and streptomycin and subsequent treatment with chloramphenicol and erythromycin, the patient died. Postmortem cultures of the lung yielded *Acinetobacter* species, *Pseudomonas aeruginosa, S. aureus,* and other *Pseudomonas* species. The lung-tissue cultures also yielded *Legionella bozemanii,* suggesting the possibility that this organism was a cause of the patient's illness.

P. aeruginosa is commonly isolated from both freshwater and seawater [23, 58]. Despite this water tropism, the organism is uncommonly reported as a cause a pneumonia after near-drowning [59–61]. The paucity of reported cases of near-drowning-associated *P. aeruginosa* pneumonia is likely due to the difficulty in differentiating pneumonia due to aspiration of the organism during near drowning from a purely nosocomial process. Furthermore, this organism as a cause of pneumonia may not be believed ''unique'' to the clinicians caring for near-drowning victims; therefore the illness may not be reported. Three of the four reported cases of this illness occurred after immersion in hot tubs, and the fourth occurred after immersion in a river. Two of the patients developed illness within 48 hours, the other two developed illness within 5 days; two of the four patients died. Physicians must consider *P. aeruginosa* in the differential diagnosis of organisms causing near-drowning-associated pneumonia, particularly during the first 5 days after admission and in the setting of near-drowning in a hot tub.

Vibrio species are common water inhabitants typically found in saltwater and brackish water, but they are also recovered from freshwater [23, 58]. While numerous cases of waterborne vibrio infection have been reported [62, 63], few of these have been pneumonias.

Four cases of near-drowning-associated pneumonia caused by *Vibrio* species have been reported in the literature [62–64]. However, the clinical details are reported in only two of these cases. A 44-year-old healthy male developed fever 4 days after a saltwater near-drowning. Both blood and sputum cultures yielded a non-*cholera Vibrio* species. The organism was also isolated from water in the region of the submersion event. The patient defervesced after three days of tobramycin therapy but went into cardiac arrest 2 days later and died [64]. Another described case contains less clinical information. A 34-year-old otherwise healthy male developed *Vibrio vulnificus* pneumonia after a saltwater near drowning. He was treated with cefoxitin and tobramycin but died 2 days after hospital admission. Unfortunately, culture and chest radiographic details were not reported [63].

Hence, while *Vibrio* species often cause illness associated with saltwater exposure, they are rarely reported as a cause of pneumonia associated with near-drowning. Nevertheless, they should be included in the differential diagnosis of pneumonia associated with near-drowning. *Vibrio* species are susceptible to many antimicrobials including tetracyclines, fluoroquinolones, most β-lactams, and trimethoprim-sulfamethoxazole [63–66].

Other aerobic gram-negative organisms. Many aerobic gram-negative bacilli have been isolated from saltwater and freshwater but have not been reported to cause pneumonia associated with near-drowning [23, 58]. However, one would expect that many of these organisms are capable of causing this illness. *Shewanella putrefaciens,* a natural inhabitant of the ocean, was implicated in one case of pneumonia after near-drowning [67]. The victim, a 56-year-old healthy male, developed pulmonary edema and respiratory failure and required mechanical ventilation. He quickly recovered and was extubated, but he developed fever 4 days after the submersion event. Sputum cultures yielded both *S. putrefaciens* and *S. aureus,* and water obtained from the saltwater near-drowning site yielded *S. putrefaciens.* Chest radiographic findings and blood culture results obtained during the patient's febrile illness were not reported, leaving the diagnosis of pneumonia difficult to confirm. *S. putrefaciens* is usually susceptible to aminoglycosides, extended-spectrum penicillins, extended-spectrum cephalosporins, and fluoroquinolones [67–69]. It is of interest that a high rate of resistance to imipenem was reported in one recent case series of *S. putrefaciens* bacteremia [68].

Some aerobic gram-negative organisms also inhabit the oropharyngeal tract in the immunocompetent and nonhospitalized host. One would expect that these organisms may cause pneumonia associated with near-drowning. However, near-drowning-associated pneumonia due to typical aerobic gram-negative orophayngeal flora has only been reported for one organism, *Neisseria mucosa,* in only one case report [70]. A 21-year-old alcohol abusing female developed bilateral pulmonary infiltrates and fever soon after admission to the hospital because of a near-drowning event in brackish water. Two sets of blood cultures performed 48 hours after admission yielded gram-negative coccobacilli, later identified as *N. mucosa.* The patient had persistent fever despite antimicrobial therapy that included tobramycin, chloramphenicol, cefotaxime, and erythromycin. She developed progressive multiorgan system failure and died on day 14 of her hospitalization. Postmortem examination failed to confirm bacterial pneumonitis. This case report is supportive, although not conclusively, of the finding that *N. mucosa* is a cause of near-drowning-associated pneumonia.

Aerobic gram-positive bacteria. Few species of aerobic gram-positive bacteria are cultured from either seawater or freshwater [23, 58]. Therefore, as one would expect, these organisms are less commonly reported as a cause of pneumonia after submersion. However, some aerobic gram-positive organisms inhabit the human oropharyngeal mucosa. These potentially pathogenic organisms may be translocated into the more-distal airways during an aspiration event. Despite this potential mechanism of infection, of the aerobic gram-positive bacteria, only *Streptococcus pneumoniae* and *S. aureus* have been reported to cause this illness.

While *S. pneumoniae* is not a typical inhabitant of freshwater or seawater, the organism commonly colonizes the upper respiratory tract. Hence, near-drowning-associated pneumonia due to this organism probably results from aspiration-induced lung damage and translocation of the bacteria to the distal pulmonary airways. Pneumococcal pneumonia has been reported as causing aspiration pneumonia [71]. Vernon et al. [72] described three cases of near-drowning-associated pneumonia due to *S. pneumoniae.* Three healthy young children were involved in these events. Two of the events occurred in freshwater (one in a river and another in a swimming pool), and one occurred in saltwater. Each child developed fever, neutropenia, and cardiopulmonary instability 12–24 hours after the near-drowning and died within 1 week. Cultures of blood and sputum yielded *S. pneumoniae* in all of the cases.

Kennedy et al. [5] reviewed 21 cases of aspiration that involved children, seven of whom developed bacterial infections. Thirteen near-drownings were included in the 21 cases. Furthermore, five of the seven infected individuals were near-drowning victims. Four of the victims had *S. pneumoniae* bacteremia (three also had *S. pneumoniae* recovered from sputum samples). This report does not describe which of the children with *S. pneumoniae* infection were near-drowning victims; however, on the basis of the information provided, at least two of them must have fallen into this category. In addition, the review does not describe chest radiographic findings or clinical details of the individual cases. Nonetheless, on the basis of the available case reports, *S. pneumoniae* appears to cause near-drowning-associated pneumonia, and empirical therapy should provide coverage for this organism in cases of pneumonia after a near-drowning event. This concept is especially true for children, as all of the reported events have involved young, healthy children.

S. aureus is culturable from both freshwater and seawater [23, 58]. The organism also frequently colonizes the nasopharyngeal tract [73]. *S. aureus* has been described as causing near-drowning-associated pneumonia in only one case [7] that is not reported in detail. The paucity of case reports of near-drowning-associated pneumonia due to this organism may represent a reporting bias, since pneumonia due to this organism in a critically ill patient may simply represent a nosocomial process. As with pneumonia due to the Enterobacteriaceae, differentiating *S. aureus* pneumonia due to near drowning vs. a nosocomial process is difficult. Therefore, clinicians may be reluctant to report such cases. Nonetheless, while *S. aureus* may represent a potential pathogen in cases of near-drowning-associated pneumonia, the issue remains unsettled.

Anaerobes. Anaerobes are not typically isolated from the aquatic environment, but they are common inhabitants of the oropharyngeal tract. Further, they are the most common pathogens causing aspiration pneumonia [74]. As near-drowning victims frequently aspirate vomitus and oropharyngeal contents, one would expect these organisms to cause pneumonia associated with near-drowning. Between 25% and 50% of gastric aspiration victims develop pulmonary infection [5, 75, 76]. It is surprising that anaerobes have not been reported as a cause of pneumonia associated with near-drowning. This notion probably reflects the difficulty in isolating these organisms in culture, as well as a reporting bias, as noted with nosocomial pathogens. Although these organisms are not described as causing near-drowning-associated pneumonia, they should still be considered a potential cause of pneumonia after an immersion event.

Fungi. Numerous potentially pathogenic fungi are ubiquitous in the environment and have been isolated from freshwater, saltwater, and contaminated water [77, 78]. Most of these organisms are typically opportunistic. However, in the setting of near-drowning, at least one—and probably two—of these organisms causes severe infection in the immunocompetent host. Damage sustained by the lung tissue from the immersion event and/or the large inoculum of organisms associated with submersion may predispose the immunocompetent host to infection with fungi. Both *Pseudallescheria boydii* and *Aspergillus* species have been reported to cause this illness [12–17, 79–82].

P. boydii (and its asexual form, *Scedosporium apiospermum*) is a ubiquitous fungus found in coastal waters and freshwater as well as in polluted or contaminated water [14, 77, 78]. Pneumonia due to this organism has been reported in several cases of near drowning [12–17, 79, 81, 82].

Most of the cases of *P. boydii* pneumonia occurred in otherwise healthy individuals who were involved in near-drowning events that occurred in sewage, polluted water, stagnant water, or muddy water. Some of the patients developed evidence of this infection within 1 week after immersion, but others manifested this illness much later. Two of the patients became ill 4–6 months after the initial near-drowning event. Nearly all of the reported cases of this illness involved dissemination of the organism, particularly to the CNS [12–17, 79, 81, 82].

The CNS manifestations usually include meningitis and brain abscesses. Another site of dissemination is the eye, with the development of endophthalmitis [16]. *P. boydii* pneumonia should be included in the differential diagnosis for any near-drowning victim who develops neurological abnormalities after a period of convalescence or develops meningitis or brain abscess. The infection should also be considered in those with evidence of meningitis or brain abscess, even if pneumonia is not suspected, as some patients have developed evidence of neurological infection before pneumonia was diagnosed [15, 17]. Cultures and stains of CSF are often negative despite the fact that multiple specimens are obtained by lumbar puncture; therefore, physicians may need to perform ventricular aspiration or brain biopsy to obtain appropriate specimens for staining and culture or treat such patients empirically [15–17, 79].

P. boydii infection is very difficult to treat, as the organism is often resistant to amphotericin B [83]. Miconazole appears to have the best in vivo activity against *P. boydii* [82, 84], and most patients have been treated with this antifungal by using both intravenous and intraventricular routes of administration (nearly all of the patients had disseminated CNS infection) [12, 14, 16, 82]. Itraconazole, fluconazole, and ketoconazole appear to have reasonable in vitro activity against this organism [85]. *P. boydii* pneumonia has been treated successfully with itraconazole, and some investigators have recommended this agent as first-line treatment for infections due to this fungus [86–88]. *P. boydii* infection has also been cured with use of ketoconazole [89]. However, most victims with this infection have died; those who recovered were treated with miconazole [82].

Aspergillus species are found in various locations including soil, seawater, polluted water, and sewage [77, 78, 80]. Therefore, one would expect this organism to be a potential cause of pneumonia associated with near-drowning. However, this fungus has rarely been reported to cause pneumonia associated with submersion. One case of invasive pulmonary aspergillosis occurred in a 27-year-old male with bronchiectasis after nearly drowning in a ditch [80]. The patient recovered after treatment with intravenous and aerosolized amphotericin B and flucytosine. The organism was not recovered from the near-drowning site. While *P. boydii* is more commonly reported to cause pneumonia associated with near drowning, *Aspergillus* is a potential cause of this illness.

Diagnosis

The diagnosis of pneumonia associated with near-drowning is difficult. Uninfected near-drowning victims may have signs, symptoms, and diagnostic findings suggestive of pneumonia. Most near-drowning victims will have pulmonary symptoms and abnormalities on chest radiographs. The radiographic abnormalities are variable but are often bilateral and diffuse [2, 7, 18, 20, 90], which can obscure other abnormalities suggestive of pulmonary infection. Some victims have fever and leukocytosis in the absence of infection [20, 90]. Critically ill victims may develop hypotension due to cardiac damage or electrolyte abnormalities, and this condition may mimic septic shock [1, 4, 90]. Cultures of sputum and specimens obtained at bronchoscopy are often difficult to interpret for any critically ill patient, and near drowning may further complicate this interpretation. Organisms growing in these cultures may represent true pathogens, airway colonization, or contamination from the environment.

Rigorous diagnosis of near-drowning-associated pneumonia is based on the presence of signs and symptoms suggestive of pneumonia, a new focal pulmonary infiltrate on a chest radiograph, or histopathologic evidence of pneumonia, as well as isolation of the organism from the blood, the airway, and water from the near-drowning location. Unfortunately, environmental cultures are rarely obtained. Some organisms, particularly the gram-negative bacilli such as *Aeromonas, C. violaceum, B. pseudomallei,* and *F. philomiragia,* are frequently isolated from the blood in cases of near-drowning-associated pneumonia. The identification of bacteremia adds strong evidence of illness and allows directed antimicrobial therapy. Therefore, victims of near-drowning should have blood for cultures drawn, particularly if they are critically ill or have evidence of infection. However, some organisms that cause pneumonia associated with near-drowning, such as fungi, are difficult to culture from the blood. In many cases of this illness, the findings necessary to make a rigorous diagnosis are lacking, and one must often rely on a compatible clinical scenario with suggestive radiographic and/or culture data.

Management

While near-drowning has been studied both prospectively and retrospectively (albeit in small studies), some aspects of management of near-drowning victims remain controversial [5, 6, 7, 91]. In particular, the management of near-drowning in relation to pneumonia remains controversial. These controversies may be difficult to settle without large, prospective trials, which would be difficult to perform.

Prophylactic antimicrobials. The use of prophylactic antibiotics is generally not recommended [1, 3, 8, 18, 19, 90]. Two retrospective reviews of near-drowning have addressed this issue, and neither showed any benefit from using prophylactic antibiotics [6, 7]. No reduction in pneumonia or mortality was noted. Furthermore, those individuals who develop pneumonia while receiving prophylactic antibiotics are often infected with resistant organisms. In addition, several reviews of aspiration did not show benefit from using prophylactic antibiotics [76, 92–94]. Some authors have recommended frequent sputum cultures and the use of antimicrobials directed at specific pathogens [6, 7, 8, 19]. However, interpretation of sputum culture results in the setting of near-drowning and critical illness is difficult; therefore, this approach should be avoided in the absence of other evidence of pulmonary infection. In their prospective evaluation of aspiration (most of the cases were near-drowning victims), Kennedy et al. [5] noted a high rate of infection. Therefore, these investigators suggest using prophylactic antibiotics in the setting of severe aspiration. Other investigators have suggested using antibiotics in the setting of near-drowning in contaminated water [1, 3, 60]. In most situations, the use of prophylactic antibiotics in the setting of near-drowning should be avoided.

Antimicrobial therapy. The decision to start antibiotic therapy for potential near-drowning-associated pneumonia is often a difficult one. Near-drowning victims often display signs and symptoms of pneumonia, even in the absence of this ailment. Many of these victims have fever and leukocytosis without other evidence of infection [20, 90]. Most have pulmonary signs and symptoms after submersion events. Chest radiographs are often difficult to interpret in this setting [2, 18, 20, 90]. As near-drowning-associated pneumonia may be particularly severe in association with some organisms [9–17], the threshold for starting empirical antibiotic therapy should be low.

Individuals who should receive antibiotics include victims who either present with or develop fever, pulmonary infiltrates, and evidence of systemic toxicity or hemodynamic instability without a clear source. Although this conservative approach results in the treatment of some individuals without infection, it will prevent clinicians from missing most cases of this potentially devastating complication of near-drowning. Most bacterial causes of near-drowning-associated pneumonia can be treated with an extended-spectrum penicillin/β-lactamase inhibitor combination. If the patient is moderately ill, the addition of an aminoglycoside should be considered. A reasonable empirical alternative for those individuals with significant penicillin allergies includes clindamycin and a fluoroquinolone.

The use of empirical antifungal therapy for near-drowning victims should generally be avoided. However, if the victim is slow to respond to antibacterial therapy, develops pneumonia several weeks after the submersion event, or develops brain abscess and/or meningitis, a diagnosis of invasive fungal disease should be aggressively pursued. The most common cause of near-drowning-associated pneumonia due to fungi, *P. boydii,* is very difficult to diagnose microbiologically and is usually not susceptible to amphotericin B. Therefore, empirical antifungal therapy is difficult to provide, even when there is a high clinical suspicion of fungal pneumonia. Regardless of empirical therapy, definitive treatment needs to be based on culture and susceptibility testing results (if available) because of the extremely broad microbial causes of this illness.

Glucocorticoids. The routine use of steroids in the setting of near-drowning should be avoided. Numerous animal and human trials conducted to study near-drowning and aspiration have not revealed any clinical benefit from using corticosteroids [6, 7, 76, 93, 95, 96]. Without a clearly defined benefit from the use of corticosteroids, they should be avoided in this setting because they have significant side effects, including immunosuppression. While cerebral edema is less common in these victims than once believed [6, 18, 91], corticosteroid therapy may be required if this complication develops.

Induced hypothermia. Another controversial therapeutic modality used in treating near-drowning victims is induced hypothermia. When hypothermia occurs during the immersion, it protects the brain from ischemic damage [97, 98]. However, when hypothermia is induced after near-drowning, it does not appear to be protective. Furthermore, hypothermia induces neutropenia [91]. Data from animal studies suggest a 50% decrease in the number of polymorphonuclear leukocytes with induced hypothermia [99]. Near-drowning victims treated with hypothermia develop significant neutropenia and are at higher risk of infection [91]. Therefore, induced hypothermia should be avoided as a treatment for near-drowning.

Bronchoalveolar lavage. The usefulness of bronchoalveolar lavage in evaluating victims of aspiration events is unclear. Whether this procedure may be useful in decreasing the risk of pneumonia after submersion is equally nebulous. The theoretical benefit of bronchoalveolar lavage for near-drowning victims includes a reduction in lung parenchymal damage from aspiration (which potentially increases the risk of pneumonia) and the removal of contaminated particulate material from the airways. One autopsy study revealed that 70% of victims had foreign particulate material in their airways [20]. This material included vomitus, mud, sand, and fragments of aquatic vegetation. Victims at particularly high risk of aspirating contaminated material include those immersed in grossly contaminated water, those drowning in shallow water, and those who display a violent agonal struggle near the bottom.

Unfortunately, parenchymal lung damage from acid aspiration occurs almost instantaneously, and bronchial secretions are buffered within minutes [92, 95, 100]. Furthermore, most experimental studies of therapeutic lavage for victims of aspiration show either no improvement or more damage [95, 101, 102]. Bronchoalveolar lavage is generally recommended for removal of large particulate matter, particularly in the setting of loss of lung volume [75, 92]. Therefore, if this complication exists in the setting of near-drowning, this procedure should be performed. Whether lavage is indicated for victims of near-drowning in contaminated water is unclear. However, bronchoalveolar lavage cannot yet be recommended as routine preventative treatment of near-drowning-associated pneumonia.

Conclusion

Pneumonia associated with near-drowning is a severe complication of a potentially devastating event. While the incidence of this infection is not known, it is certainly not a rare event. The risk factors for developing this illness after near-drowning are not clearly defined because of the paucity of studies addressing this issue. However, if clinicians understand the pathophysiology of near-drowning, they can predict likely risk factors for the development of pneumonia. It can be intuitively expected that aspiration of the aquatic environment (as opposed to ''dry'' near-drowning) increases the risk of pneumonia, particularly when the water is contaminated. The aspiration of gastric contents probably also increases a victim's risk of developing pneumonia because of the resultant pulmonary damage. Other factors, such as the temperature and chemical composition of the water, may also impact on risk. However, until the risk factors for near-drowning-associated pneumonia are prospectively studied, risk assessment is difficult.

The organisms that cause pneumonia after submersion either originate from the aquatic environment or from the oropharyngeal airway after an aspiration event. A wide variety of bacterial organisms and some fungal pathogens can cause this illness. The pathogens suspected of causing illness can be postulated on the basis of the environment of the near-drowning and the character of the presenting illness. While prophylactic antibiotics are not recommended for near-drowning victims, the threshold for using empirical antibiotics to treat the probable pathogens should be low. Avoidance of therapies for near-drowning that have not been proven beneficial and are potentially harmful, such as corticosteroids and hypothermia, is also recommended.

References

1. Modell JH. Drowning. N Engl J Med **1993**;328:253–6.
2. Olshaker JS. Near drowning. Emerg Med Clin North Am **1992**;10:339–50.
3. Levin DL, Morriss FC, Toro LO, Brink LW, Turner GR. Drowning and near drowning. Pediatr Clin North Am **1993**;40:321–36.
4. Hoff BH. Multisystem failure: a review with special reference to drowning. Crit Care Med **1979**;7:310–20.
5. Kennedy GA, Kanter RK, Weiner LB, Tompkins JM. Can early bacterial complications of aspiration with respiratory failure be predicted? Pediatric Emergency Care **1992**;8:123–5.
6. Oakes DD, Sherck JP, Maloney JR, Charters AC III. Prognosis and management of victims of near-drowning. J Trauma **1982**;22:544–9.
7. Modell JH, Graves SA, Ketover A. Clinical course of 91 consecutive near-drowning victims. Chest **1976**;70:231–8.
8. Orlowski JP. Drowning, near-drowning and ice-water submersions. Pediatr Clin North Am **1987**;34:75–92.
9. Ender PT, Dolan MJ, Dolan D, Farmer JC, Melcher GP. Near-drowning-associated *Aeromonas* pneumonia. J Emerg Med **1996**;14:737–41.
10. Greenawald KA, Nash G, Foley FD. Acute systemic melioidosis: autopsy findings in four patients. Am J Clin Pathol **1969**;52:188–98.
11. Achana V, Silpapojakul K, Thininta W, Kalnaowakul S. Acute *Pseudomonas pseudomallei* pneumonia and septicemia following aspiration of contaminated water: a case report. Southeast Asian Trop Med Public Health **1985**;16:500–4.
12. Travis LB, Roberts GD, Wilson WR. Clinical significance of *Pseudallescheria boydii:* a review of 10 years' experience. Mayo Clin Proc **1985**;60:531–7.
13. Durieu I, Parent M, Ajana F, et al. *Monosporium apiospermum* meningoencephalitis: a clinico-pathological case. J Neurol Neurosurg Psychiatry **1991**;54:731–3.
14. Fisher JF, Shadomy S, Teabeaut JR, et al. Near-drowning complicated by brain abscess due to *Petriellidium boydii.* Arch Neurol **1982**;39:511–3.
15. Dubeau F, Roy LE, Allard J, et al. Brain abscess due to *Petriellidium boydii.* Can J Neurol Sci **1984**;11:395–8.
16. Meadow WL, Tipple MA, Rippon JW. Endophthalmitis caused by *Petriellidium boydii.* Am J Dis Child **1981**;135:378–80.
17. Kershaw P, Freeman R, Templeton D, et al. *Pseudallescheria boydii* infection of the central nervous system. Arch Neurol **1990**;47:468–72.
18. Gonzalez-Rothi RJ. Near drowning: consensus and controversies in pulmonary and cerebral resuscitation. Heart Lung **1987**;16:474–82.
19. Modell JH. Biology of drowning. Annu Rev Med **1978**;29:1–8.
20. Fuller RH. The clinical pathology of human near-drowning. Proc R Soc Med **1963**;56:33–8.
21. Modell JH, Davis JH. Electrolyte changes in human drowning victims. Anesthesiology **1969**;30:414–20.
22. Rheinheimer G. The influence of physical and chemical factors on aquatic micro-organisms. In: Rheinheimer G. Aquatic microbiology. 3rd ed. New York: John Wiley & Sons, **1985**:95–117.
23. Sims JK, Enomoto PI, Frankel RI, Wong LMF. Marine bacteria complicating seawater near-drowning and marine wounds: a hypothesis. Ann Emerg Med **1983**;12:212–6.
24. Modell JH, Gaub M, Moya F, Vestal B, Swarz H. Physiologic effects of near drowning with chlorinated fresh water, distilled water and isotonic saline. Anesthesiology **1966**;27:33–41.
25. George DL. Epidemiology of nosocomial pneumonia in the intensive care unit patients. Clin Chest Med **1995**;16:29–44.
26. Reines HD, Cook FV. Pneumonia and bacteremia due to *Aeromonas hydrophila.* Chest **1981**;80:264–7.
27. Baddour LM, Baselski VS. Pneumonia due to *Aeromonas hydrophila*-complex: epidemiologic, clinical, and microbiologic features. South Med J **1988**;81:461–3.
28. Goncalves JR, Brum G, Fernandes A, Biscaia I, Correia MJS, Bastardo J. *Aeromonas hydrophila* fulminant pneumonia in a fit young man. Thorax **1992**;47:482–3.
29. Mani S, Sadigh M, Andriole VT. Clinical spectrum of *Aeromonas hydrophila* infections: report of 11 cases in a community hospital and review. Infectious Diseases in Clinical Practice **1995**;4:79–86.
30. Lecler O, Pourrait JL, Hoang P, Fournier JL, Cupa M. Infection pulmonaire a *Aeromonas hydrophila* après noyade en piscine. Cah Anesthesiol **1990**;38:435–6.
31. Genoni L, Domenighetti G. Beinahe-Ertrinken beim Erwachsenen: günstiger Verlauf nach 20 minütiger Submersionszeit. Schweiz Med Wochenschr J **1982**;112:867–70.
32. Outin HD, Chatelin A, Ronco E, et al. Septicémies à *Aeromonas hydrophila:* trios observations dont une avec médiastinite. An Med Interne (Paris) **1984**;135:287–90.
33. Gaussorgues P, Bachmann P, Tigaud S, et al. Abcès pulmonaire à *Aeromonas hydrophila:* complication de noyade en eau douce. Ann Med Interne (Paris) **1987**;138:666–7.
34. Jones BL, Wilcox MH. Aeromonas infections and their treatment. J Antimicrob Chemother **1995**;35:453–61.
35. Howe C, Sampath A, Spotnitz M. The pseudomallei group: a review. J Infect Dis **1971**;124:598–606.
36. Strauss JM, Groves MG, Mariappan M, Ellison DW. Melioidosis in Malaysia: II. Distribution of *Pseudomonas pseudomallei* in soil and surface water. Am J Trop Med Hyg **1969**;18:698–702.
37. Nachiangmai N, Patamasucon P, Tipayamonthein B, Kongpon A, Nakaviroj S. *Pseudomonas pseudomallei* in southern Thailand. Southeast Asian J Trop Med Public Health **1985**;16:83–7.
38. Lee N, Wu J-L, Lee C-H, Tsai W-C. *Pseudomonas pseudomallei* infection from drowning: the first reported case in Taiwan. J Clin Microbiol **1985**;22:352–4.

39. Yamamoto T, Naigowit P, Dejsirilert S, et al. In vitro susceptibilities of *Pseudomonas pseudomallei* to 27 antimicrobial agents. Antimicrob Agents Chemother **1990**;34:2027–9.
40. Hall WH, Manion RE. Antibiotic susceptibility of *Pseudomonas pseudomallei.* Antimicrob Agents Chemother **1973**;4:193–5.
41. Anonymous. The choice of antibacterial drugs. Medical Lett Drugs Ther **1996**;38:25–34.
42. Macher AM, Casale TB, Fauci AS. Chronic granulomatous disease of childhood and *Chromobacterium violaceum* infections in the southeastern United States. Ann Intern Med **1982**;97:51–5.
43. Ponte R, Jenkins SG. Fatal *Chromobacterium violaceum* infections associated with exposure to stagnant waters. Pediatr Infect Dis J **1992**;11: 583–6.
44. Centers for Disease Control. Chromobacteriosis—Florida. MMWR **1981**;29:613–5.
45. Myers J, Ragasa DA, Eisele C. *Chromobacterium violaceum* septicemia in New Jersey. J Med Soc NJ **1982**;79:213–4.
46. Starr AJ, Cribbett LS, Poklepovic J, Friedman H, Ruffolo EH. *Chromobacterium violaceum* presenting as a surgical emergency. South Med J **1981**;74:1137–9.
47. Aldridge KE, Valainis GT, Sanders CV. Comparison of the in vitro activity of ciprofloxacin and 24 other antimicrobial agents against clinical strains of *Chromobacterium violaceum.* Diagn Microbiol Infect Dis **1988**;10:31–9.
48. Wenger JD, Hollis DG, Weaver RE, et al. Infection caused by *Francisella philomiragia* (formerly *Yersinia philomiragia*): a newly recognized human pathogen. Ann Intern Med **1989**;110:888–92.
49. Greco D, Allegrini G, Tizzi T, Ninu E, Lamanna A, Luzi S. A waterborne tularemia outbreak. Eur J Epidemiol **1987**;3:35–8.
50. Jensen WI, Owen CR, Jellison WL. *Yersinia philomiragia* sp. n., a new member of the *Pasteurella* group of bacteria, naturally pathogenic for the muskrat (*Ondatra zibethica*). J Bacteriol **1969**;100:1237–41.
51. Rivers JF, Orr G, Lee HA. Drowning. Its clinical sequelae and management. Br Med J **1970**;2:157–61.
52. Barr AM, Taylor M. A case of drowning. Anaesthesia **1976**;31:651–7.
53. Fliermans CB, Cherry WB, Orrison LH, Smith SJ, Tison DL, Pope DH. Ecological distribution of *Legionella pneumophila.* Appl Environ Microbiol **1981**;41:9–16.
54. Fang G-D, Yu VL, Vickers RM. Disease due to the Legionellaceae (other than *Legionella pneumophila*): historical, microbiological, clinical, and epidemiologic review. Medicine (Baltimore) **1989**;68:116–32.
55. Shiota R, Takeshita K, Yamamoto K, Imada K, Yabuuchi E, Wang L. *Legionella pneumophila* serogroup 3 isolated from a patient of pneumonia developed after drowning in bathtub of a hot spring spa [in Japanese]. Kansenshogaku Zasshi **1995**;69:1356–64.
56. Thomason BM, Harris PP, Hicklin MD, Blackmon JA, Moss CW, Matthews F. A *Legionella*-like bacterium related to WIGA in a fatal case of pneumonia. Ann Intern Med **1979**;91:673–6.
57. Cordes LG, Wilkinson HW, Gorman GW, Fikes BJ, Fraser DW. Atypical Legionella-like organisms: fastidious water-associated bacteria pathogenic for man. Lancet **1979**;2:927–30.
58. Auerbach PS, Yajko DM, Nassos PS, Kizer KW, Morris JA Jr, Hadley WK. Bacteriology of the freshwater environment: implications for clinical therapy. Ann Emerg Med **1987**;16:1016–22.
59. Rose HD, Franson TR, Sheth NK, Chusid MJ, Macher AM, Zeirdt CH. *Pseudomonas* pneumonia associated with use of a home whirlpool spa. JAMA **1983**;250:2027–9.
60. Tron VA, Baldwin VJ, Pirie GE. Hot tub drownings. Pediatrics **1985**; 75:789–90.
61. Modell JH. Hospital therapy. In: The pathophysiology and treatment of drowning and near-drowning. Springfield, Illinois: Charles C. Thomas, **1971**:95–113.
62. Hlady WG, Klontz KC. The epidemiology of *Vibrio* infections in Florida, 1981–1993. J Infect Dis **1996**;173:1176–83.
63. Chuang Y-C, Yuan C-Y, Liu C-Y, Lan C-K, Huang AH-M. *Vibrio vulnificus* infection in Taiwan: report of 28 cases and review of clinical manifestations and treatment. Clin Infect Dis **1992**;15:271–6.
64. Kelly MT, Avery DM. Lactose-positive *Vibrio* in seawater: a cause of pneumonia and septicemia in a drowning victim. J Clin Microbiol **1980**;11:278–80.
65. Clark RB. Antibiotic susceptibilities of the Vibrionaceae to meropenem and other antimicrobial agents. Diagn Microbiol Infect Dis **1992**;15:453–5.
66. Joseph SW, DeBell RM, Brown WP. In vitro response to chloramphenicol, tetracycline, ampicillin, gentamicin, and beta-lactamase production by halophilic vibrios from human and environmental sources. Antimicrob Agents Chemother **1978**;13:244–8.
67. Rosenthal SL, Zuger JH, Apollo E. Respiratory colonization with *Pseudomonas putrefaciens* after near-drowning in salt water. Am J Clin Pathol **1975**;64:382–4.
68. Brink AJ, van Straten A, van Rensburg AJ. *Shewanella* (*Pseudomonas*) *putrefaciens* bacteremia. Clin Infect Dis **1995**;20:1327–32.
69. Gilardi GL. Antimicrobial susceptibility as a diagnostic aid in the identification of nonfermenting gram-negative bacteria. Appl Microbiol **1971**;22:821–3.
70. Manser TJ, Warner JF. *Neisseria mucosus* septicemia after near-drowning. South Med J **1987**;80:1323–4.
71. Kanter RK, Carroll JL. Early pneumococcal sepsis after pulmonary aspiration and the adult respiratory distress syndrome. Crit Care Med **1983**; 11:906–7.
72. Vernon DD, Banner W Jr, Cantwell GP, Holzman BH, Bolte RG, Dean JM. *Streptococcus pneumoniae* bacteremia associated with near-drowning. Crit Care Med **1990**;18:1175–6.
73. Fekety FR Jr. The epidemiology and prevention of Staphylococcal infection. Medicine (Baltimore) **1964**;43:593–613.
74. Bartlett JG, Gorbach SL, Finegold SM. The bacteriology of aspiration pneumonia. Am J Med **1974**;56:202–7.
75. Arms RA, Dines DE, Tinstman TC. Aspiration pneumonia. Chest **1974**; 65:136–9.
76. Bynum LJ, Pierce AK. Pulmonary aspiration of gastric contents. Am Rev Respir Dis **1976**;114:1129–36.
77. Dabrowa N, Landau JW, Newcomer VD, Plunkett OA. A survey of tide-washed coastal areas of Southern California for fungi potentially pathogenic to man. Mycopathologia **1964**;24:136–50.
78. Cooke WB, Kabler P. Isolation of potentially pathogenic fungi from polluted water and sewage. Public Health Rep **1955**;70:689–94.
79. Gari M, Fruit J, Rousseaux P, et al. *Scedosporium* (*Monosporium*) *apiospermum:* multiple brain abscesses. Sabouraudia **1985**;23:371–6.
80. Vieira DF, Van Saene HKF, Miranda DR. Invasive pulmonary aspergillosis after near-drowning. Intensive Care Med **1984**;10:203–4.
81. van der Vliet JA, Tidow G, Kootstra G, et al. Transplantation of contaminated organs. Br J Surg **1980**;67:596–8.
82. Dworzack DL, Clark RB, Padgitt PJ. New causes of pneumonia, meningitis, and disseminated infections associated with immersion. Infect Dis Clin North Am **1987**;1:615–33.
83. Lutwick LI, Galgiani JN, Johnson RH, Stevens DA. Visceral fungal infections due to Petrellidium boydii (Allescheria boydii): in vitro drug sensitivity studies. Am J Med **1976**;61:632–40.
84. Lutwick LI, Rytel MW, Yañez JP, Galgiani JN, Stevens DA. Deep infections from *Petriellidium boydii* treated with miconazole. JAMA **1979**; 241:272–3.
85. Walsh TJ, Peter J, McGough DA, Fothergill AW, Rinaldi MG, Pizzo PA. Activities of amphotericin B and antifungal azoles alone and in combination against *Pseudallescheria boydii.* Antimicrob Agents Chemother **1995**;39:1361–4.
86. Goldberg SL, Geha DJ, Marshall WF, Inwards DJ, Hoagland HC. Successful treatment of simultaneous pulmonary *Pseudallescheria boydii* and *Aspergillus terreus* infection with oral itraconazole. Clin Infect Dis **1993**;16:803–5.

87. Martino R, Nomdedéu J, Altes A, et al. Successful bone marrow transplantation in patients with previous invasive fungal infections: report of four cases. Bone Marrow Transplant **1994**;13:265–9.
88. Anonymous. Systemic antifungal drugs. Med Let Drugs Ther **1996**;38:10–2.
89. Galgiani JN, Stevens DA, Graybill JR, Stevens DL, Tillinghast AJ, Levine HB. *Pseudallescheria boydii* infections treated with ketoconazole: clinical evaluations of seven patients and *in vitro* susceptibility results. Chest **1984**;86:219–24.
90. Fiser DH. Near-drowning. Pediatr Rev **1993**;14:148–51.
91. Bohn DJ, Biggar WD, Smith CR, Conn AW, Barker GA. Influence of hypothermia, barbiturate therapy, and intracranial pressure monitoring on morbidity and mortality after near-drowning. Crit Care Med **1986**;14:529–34.
92. Wynne JW, Modell JH. Respiratory aspiration of stomach contents. Ann Intern Med **1977**;87:466–74.
93. Cameron JL, Mitchell WH, Zuidema GD. Aspiration pneumonia: clinical outcome following documented aspiration. Arch Surg **1973**;106:49–52.
94. Lewis RT, Burgess JH, Hampson LG. Cardiorespiratory studies in critical illness: changes in aspiration pneumonitis. Arch Surg **1971**;103:335–40.
95. Awe WC, Fletcher WS, Jacob SW. The pathophysiology of aspiration pneumonitis. Surgery **1966**;60:232–9.
96. Calderwood HW, Modell JH, Ruiz BC. The ineffectiveness of steroid therapy for treatment of fresh-water near-drowning. Anesthesiology **1975**;43:642–50.
97. Young RSK, Zalneraitis EL, Dooling EC. Neurological outcome in cold water drowning. JAMA **1980**;244:1233–5.
98. Sekar TS, MacDonnell KF, Namsirikul P, Herman RS. Survival after prolonged submersion in cold water without neurologic sequelae: report of two cases. Arch Intern Med **1980**;140:775–9.
99. Biggar WD, Bohn D, Kent G. Neutrophil circulation and release from bone marrow during hypothermia. Infect Immun **1983**;40:708–12.
100. Hamelberg W, Bosomworth PP. Aspiration pneumonitis: experimental studies and clinical observations. Anaesthesia and Analgesia **1964**;43:669–77.
101. Bannister WK, Sattilaro AJ, Otis RD. Therapeutic aspects of aspiration pneumonitis in experimental animals. Anesthesiology **1961**;22:440–3.
102. Wamberg K, Zeskov B. Experimental studies on the course and treatment of aspiration pneumonia. Anesth Analg **1966**;45:230–6.